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JUL 30 1917
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THE MEDICAL JOURNAL OF AUSTRALIA

(With which "The Australasian Medical Gazette," and "The Australian Medical Journal" are incorporated.)

The Journal of the Australian Branches of the British Medical Association.

VOL. I.—4TH YEAR—NO. 26. SYDNEY: SATURDAY, JUNE 30, 1917. PRICE 6D.

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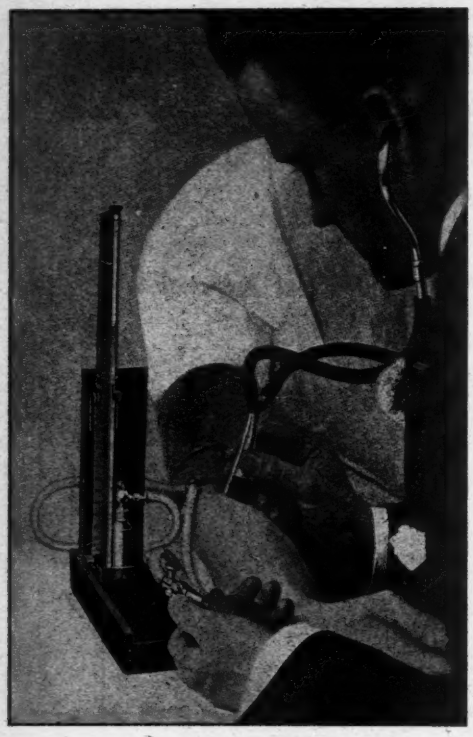

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TO THE MEDICAL PROFESSION.

We beg to state that we have received a limited number of the Ephemeris Pharmacologica for 1917 issued by Messrs. Oppenheimer, Son & Co., Ltd., London, and shall be glad to hear from any member of the Medical Profession who has not received one, and who wishes a copy, which will be sent post free.

It is a matter of regret that the necessity of keeping this Medical Diary and Visiting List a suitable size for the pocket, renders it impossible to incorporate more than a terse synopsis of the more recent publications relating to drugs. We venture to hope, however, that the 1917 edition will meet with an equally cordial reception as previous editions, and that members of the Profession will continue to favour us with suggestions for the improvement of this miniature publication.

Yours faithfully,

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THE MEDICAL JOURNAL OF AUSTRALIA.

VOL. I.—4TH YEAR. SYDNEY: SATURDAY, JUNE 30, 1917. No. 26.

DR. ERIC PRITCHARD'S THEORY AND TREATMENT OF RICKETS.[1]

By **W. F. Litchfield, M.B. (Sydney),**

Honorary Assistant Physician, Royal Alexandra Hospital for Children, Sydney; Honorary Assistant Pathologist, Royal Prince Alfred Hospital, Sydney.

In an article in the *British Journal of Children's Diseases*, of October, 1916, Dr. Eric Pritchard has elaborated a new theory of the causation of rickets, and has outlined his treatment of the disease. Dr. Pritchard's theory of the causation of rickets is that it is primarily due to the infant taking an excess of nutrient material, in other words, over-feeding, and the symptoms arise by the calling into play of certain protective mechanisms by the organism. By excess of food is meant a relative excess, and not necessarily an excess judged by ordinary standards; for instance, "if a child is kept in a hot, stuffy room and is wrapped in a multiplicity of clothes, and is seldom taken out of doors and given no opportunity for muscular exercise, he will create no demand for food, and any dietary, however small, will be relatively excessive." The chief resources at the disposal of the organism for dealing with excess of nutritive material are:—

(1) The storing up of food reserve, for instance, as glycogen and fat. The visible expression of this would be the child growing fat.

(2) The combustion of the surplus food into the end products of carbonic acid gas, urea and water. The evidence of this excessive combustion would be sweating, vascular dilatation of the capillaries of the face and possibly disturbances of the heat regulating mechanism.

(3) The short-circuiting of the processes by combustion or oxidation, with the production and absorption into the blood of acid bodies, such as lactic, diacetic, glycuronic, β-oxybutyric and certain other organic acids. Such acid bodies are not allowed to remain in the body without neutralization, and in this way the reserves of carbonates in the blood are drawn upon, ammonia is extracted from protein sources, while calcium, sodium, magnesium and iron are withdrawn or withheld from important organic combinations. The term acidosis covers the injuries inflicted on the organism in this way. The symptoms by which an acidosis is recognized are hyperpyrexia, cyclic vomiting, anæmia, a variety of nervous symptoms, demineralization of bone and a general disturbance of function. According to the author, the enlargement of the epiphyses of the long bones is due to a compensatory activity of the blood-forming organs to combat the anæmia. In any particular case of rickets it is impossible to predict the precise combination of methods adopted to dispose of excess of foodstuffs, but we could predict with complete confidence that the child would, if he could lay up stores of fat and glycogen, at first show evidence of excessive combustion by sweating and vascular dilatation of the superficial capillaries of the face and other exposed parts, and possibly by disturbances of the heat-regulating centres, and then of sub-oxidation, with the symptoms of an acidosis, with enlarged epiphyses and demineralization of bone, and finally of acyanotic hyperpnœa, with other serious nervous manifestations.

The treatment adopted by Dr. Pritchard amounts to a restriction of diet, an endeavour to create a demand for food by means such as massage, resistance exercises, cold douches and open-air treatment; the exhibition of large doses of alkali to counteract the acidosis; carbonate of iron for the anæmia, and phosphorated cod liver oil (1 in 10,000) to build up the nervous system. He claims good results from this method of treatment.

This is as fair an account of Dr. Pritchard's views as I can give in a short space. It should be noted at once that whereas the older theories on the causation of rickets assumed a deficiency of fat and protein in the food, Dr. Pritchard claims there is nothing of the sort, but an actual excess of food and overstimulation of metabolism fore-running and accounting for the disease.

I know of only one other authority who has expressed similar views. I refer to Ad. Czerny. He holds that hyper-nutrition in infants is followed by nutritional failure, which is accompanied by an acidosis, and that the symptoms of rickets follow. Many other writers have urged the dangers of overfeeding, but more on account of the digestion than the nutrition. The part played by acidosis in children's ailments, apart from rickets, has received increasing attention lately. Frequent references have been made to the ammoniacal diaper, hyperpnœa in gastro-enteritis, and to cyclic vomiting, as evidence of the occurrence of acidosis in the disorders of children.

For some years I have held similar views to those expressed by Dr. Pritchard, and I wish to give his theories and method of treatment a general support. In Australia rickets is less frequent and less severe than it is in England and some other countries. Enlarged epiphyses and the bony deformities of rickets are not common here. If we see in this country a well-marked case of hypoplastic teeth we can say with every assurance that the victim is an immigrant. Mild cases and ones of moderate severity, however, are frequently seen, and some of them are very distressing, and the cause of prolonged ill-health. The usual history is that the child was bottle fed, and got on very well up to the sixth or seventh month, then began to be restless and peevish, capricious in his appetite, became subject to attacks of diarrhœa and bronchitis and became weak

[1] Read at a Meeting of the New South Wales Branch of the British Medical Association on May 25, 1917.

and soft and pale. The usual symptoms are sweating, with miliarious rashes, an extraordinary restlessness at night, flabby subcutaneous tissues, soft, slack and weak muscles, anæmia, an uncertain appetite, irregular action of the bowels and a tendency to bronchial catarrh. The teeth are usually late in coming and the fontanelles late in closing. There may be some beading of the ribs, but this is seldom pronounced. Some of the borderland cases present these symptoms only in a mild form. Whatever is the nature of rickets, the study of it should be easier in Australia than in England, owing to the greater opportunity of seeing the disease in its early and milder stages.

As a supplement and support to Dr. Pritchard's views, I would like to state briefly how I have worked the matter out for myself. I think that at first the child, who ultimately developes rickets, is not only over-fed, but becomes over-nourished. He becomes not only fat, but overgrows in muscle and bone as well. And the muscle is good muscle and the bone good bone. But the organism cannot stand this rapid growth too long, and at the age from six to nine months fatigue begins to show itself. Much could be said about fatigue, but I want to say only this, that when fatigue occurs in an organ or a set of organs in the body, it soon manifests itself in a general disturbance of the functions of the organism. The first effect of fatigue in the case we are considering is an acidosis from the incomplete oxidation of the products of digestion. The immediate effect of the acidosis is a great stimulation of the functional activities of the body; the child becomes restless, the respiration increases, the skin acts freely and the kidney secretion amplifies. This is the beginning of rickets. The effect of these efforts of the organism are twofold; one to increase the oxidation of the partly-burned products and the other to get rid of the acids through the skin, lungs and kidneys. If the case were caught here, as it seldom is, and treated by a judicious restriction and regulation of the diet and an improvement in the surroundings and habits of the child, the disease would not progress. Unfortunately, the process usually goes on; the increased combustion calls for more food, and the beflogged organs become more distressed, the fatigue increases, the acidosis becomes more pronounced and the functions of the body depressed, and the symptoms and manifestations of rickets already enumerated appear. Amongst other things the appetite becomes depressed, and here we see an effort of the organism to save itself. If at this stage a policy of starvation or restriction of the diet were adopted, the disease could be cut short; but, as a rule, the child is coaxed or forced to take more food than it can properly deal with, and the fatigue and acidosis become chronic, or perhaps it would be safer to say chronically intermittent. Moreover, a vicious circle is established, for as soon as some relief to the fatigue is obtained by voluntary starvation the acid bodies again whip up the flagging metabolism and keep the process going. The capricious appetite in rickets is easily understood on this supposition. There seems to me an undoubted intermittence of the symptoms in rickets. Improvements and relapses occur frequently; these, I believe, are due to the more or less successful efforts of the organism in the ways already mentioned, namely, the increased combustion, the increased elimination of acid bodies, and, finally, the voluntary starvation, to right itself. It is interesting to contrast marasmus and rickets. In the former there is no intermission, the disease progresses in a steady course of failing nutrition to a fatal end. Some years ago Adalbert Czerny, Professor of Pædiatrics at Breslau, advanced the opinion that marasmus or infantile atrophy was due to an acidosis. To quote Benedict: "He regards the disease as a chronic nutritive disturbance produced by an acid intoxication from hyperacidity of the organism (acidosis), which necessitates a greater activity, to which it is unequal, on account of the toxic effects of the circulating acids. The acidosis arises from improper feeding, especially a surfeit of food." If this view be correct, then it would appear that marasmus and rickets have much the same origin, but that in the former there is no respite, while in the latter the organism is capable of making efforts to right itself. Dr. Pritchard's explanation of the enlargement of the epiphyseal expansions of the long bones in rickets being due to a hæmogenetic reaction can hardly be accepted, for there is no evidence that the hyperplasia that occurs in these regions is in any way connected with the formation of blood. Some other explanation of the phenomenon must be forthcoming; at present I do not offer one.

Now how does this theory accord with the older theories and established facts about rickets?

Some years ago Finley (*British Medical Journal*) brought forward incontestible evidence that lack of exercise was an important factor in the production of rickets. Lack of exercise with ordinary methods of feeding must soon lead to a relative hyper-nutrition, and, further, insufficient exercise must encourage an acidosis, for combustion would be deficient and elimination of acid products minimized.

Breast-fed children rarely develope rickets. There are exceptions, but we may neglect them at present. Breast-fed children are not usually overfed. Again, breast milk is an alkaline fluid and, further, being physiologicaly right, it does not stimulate the organism to excessive demands. Finally, the suckled child gets opportunities for exercise which the bottle baby does not.

One of the oldest theories of the causation of the bony deformities of rickets was that the mineral constituents of bone were dissolved out or prevented from deposition by lactic acid. This theory was abandoned because examination of the blood seemed to show that the alkalinity of the blood was not less in rickets than in health. Recent researches, however, have shown that the old methods of testing the reaction of the blood were quite unreliable, and it still remains to be shown that the acid content of the blood is not increased in rickets.

Rickets is more prevalent in cold than in warm climates. Cold weather is a stimulant to metabolism, and apt to lead to excessive alimentary demands. Moreover, it must interfere with the free actions of the skin, and also lessen the opportunities for

exercise, especially amongst the poor in overcrowded centres.

Finally, a word with regard to treatment. I look upon the disease as one essentially of fatigue resulting from overstrain of the organs of assimilation and metabolism, and resulting in an acidosis. The organs concerned should be rested by a restriction of the diet, and the acidosis must be got rid of by the use of alkalies, open-air life and exercise. I usually order the parents to restrict the feeding to three meals a day, with nothing but water in between, with the additional instruction, if the child shows a reluctance for food at any of its meals not to force it to eat. I recommend a mixed diet, arranged according to the child's age, limiting but not eliminating the fat. I prescribe a mixture containing bicarbonate of soda, potassium citrate and carbonate of magnesia. I also order the surface of the body to be well rubbed with a little cocoa-butter once or twice a day. Open-air treatment and the opportunity for exercise are also insisted on. Other treatment is symptomatic. Small doses of cod liver oil and Parrish's food is often of use, and bronchitis and derangement of the bowels may require special measures. This method of treatment in my hands has been successful. The first thing to be noticed is that the child becomes more restful, then the sweating becomes less, and soon the tone of the muscles improves and the subcutaneous tissues become firmer, and the child takes a more normal interest in his surroundings. In the severer cases a rapid restoration to health must not be expected; the fatigued tissues take time to recover, and any indiscretion or intercurrent illness may retard the progress.

Reports of Cases.

A CASE OF ANÆMIA OF "PERNICIOUS" TYPE IN AN INFANT.[1]

By **Burton Bradley, M.B., Ch.M. (Syd.), D.P.H. (Lond.),**
Sydney.

Although the exact pigeon-holing of the case to be described may not be possible, and I have a very open mind as to its relation to true Addisonian anæmia of adults, yet there seem to me to be a number of points of interest in this case from the point of view of diagnosis and prognosis, and also because the consideration of such a case illustrates some of the difficulties of the clinical pathologist.

The patient, E.M.H., was admitted on the 20th November, 1916, under Dr. Van Someren, to the Royal North Shore Hospital. The mother brought her to the Hospital because of her colour and drowsiness.

Previous History.—The child was born 11½ months previously. The confinement was normal, and the child born healthy, weighing 7¼ lbs. She was partly breast fed until three months old. Glaxo was also given. She had "gastritis" after the first month and Glaxo was stopped, and condensed milk was given for six months, and after this Nestlé's food until the date of admission. She was, except for this "gastritis," which apparently cleared up soon, quite well until she was seven months old, when she had gastro-enteritis, and the stools were slightly streaked with blood. This improved, and the stools, according to the mother, were normal at 11½ months, when she was brought to Hospital owing to the drowsiness, loss of appetite and poor colour.

[1] Read at a Meeting of the New South Wales Branch of the British Medical Association on May 25, 1917.

Family History.—The father is a painter, and is reported as a healthy man. The mother is a healthy-looking woman of about 30. She states she had anæmia, giddiness and fainting attacks as a girl of 17, and chronic throat trouble, and that up to the age of 21 she had kidney trouble, pains in the back and "septic" urine. There is a doubtful history of a miscarriage previous to the pregnancy after which the present child was born. There are no other children.

After the child was found to be anæmic, I did a blood count on the mother, which showed her blood to be normal. Recently I applied the complement deviation test to her blood with a negative result.

Condition on Admission.—When the child was admitted it was a pale lemon colour, and lay still, with her arms half extended and hands supinated. She made little effort to move, though capable of doing so, and was very sleepy. There was puffiness of the face and swelling around the ankles. The abdomen was soft. The spleen was palpable an inch below the costal margin anteriorly, and the lower edge of the liver was felt ½ to 1 inch below the costal margin, both in the mid-line and to the right side. There was a systolic murmur heard over the mitral, pulmonary and aortic areas, but not propagated in any special direction. A venous hum was present in the root of the neck. The knee-jerks were present, and the plantar response was flexor. The pupils were equal, and reacted to light. On admission the temperature was 38.3° C. (101° F.), and the pulse-rate 136.

The urine had a heavy cloud of albumin on boiling, but contained no sugar and no pus. Epithelial cells were present in considerable numbers. There were no crystals and no blood. One doubtful cast was noted.

Apart from the anæmia, there were no very special features of her condition while she was in Hospital, except that there was at times diarrhœa, with green stools containing mucus and curds, and at times pus, and periods of pyrexia up to 38.9° C. (102° F.), apparently associated with exacerbations of the diarrhœa.

Both the fever and the diarrhœa vanished as the case progressed to improvement, but I do not feel convinced that they bear any very special relation to the anæmia. When the child was admitted diarrhœa, etc., was not present, and for several days the temperature was low. A tendency to bowel disturbance is so common in children, and may depend on so very many factors, that I think it would be unwise to attribute, as some have, a causative function to it in such cases. The case was first treated with *liquor ferri perchlor. Liquor Donovani* was exhibited on 28th November, but was suspended on 2nd December, when a quinine mixture was given and calomel as required. Subsequently santonin and *filix mas* were given with the idea of excluding a worm infection. Neither worms nor ova were found.

On the 10th December, *liquor arsenicalis* was given in a dose of 0.06 c.cm. (min. i.) three times a day, and increased gradually until on the 28th January, 1917, she was taking 0.48 c.cm. three times a day. Improvement set in after the arsenic, and on the 14th December she looked much better, and was brighter. From this time she improved, until, on leaving the Hospital, she appeared practically a normal, bright little girl.

The Blood Condition.—On the 23rd November, 1916, three days after admission, a full blood count was made, with the following result:—

Red Cells	1,600,000
Hæmoglobin	30%
Leucocytes	14,000
Colour Index less than	1.0

There was marked poikilocytosis, anisocytosis and polychromatophilia. Many microcytes and macrocytes were present. There were considerable numbers of nucleated cells, some of which were undoubtedly megaloblasts.

Differential count:—

Polymorpho-nuclear Cells	37%
Mononuclear Cells	51%
Eosinophile Cells	3%
Large Mononuclear Cells	4%
Transitional Cells	5%

On the 3rd December, 1916, the count was:—

Red Cells (dupl.)	1,300,000
Hæmoglobin	25%
Leucocytes	8,500
Colour Index	1.0

There was marked poikilocytosis and anisocytosis. Polychromatophilia was present. There were many nucleated red cells, some of which are undoubtedly megaloblasts.

Differential count:—

Polymorpho-nuclear Cells	39%
Mononuclear Cells	54%
Eosinophile Cells	6%
Basophile Cells	1%

On the 10th December, 1916, the count was:—

Red Cells (dupl.)	1,600,000
Hæmoglobin	35%
Leucocytes (dupl.)	37,000
Colour Index	1.0

Differential count (300 cells counted):—

Polymorpho-nuclear Cells	65%
Mononuclear Cells	24%
Eosinophile Cells	5%
Basophile Cells	1%
Unclassifiable Cells	5%

There were plentiful nucleated red cells, chiefly megaloblasts. Polychromatophilia was present. Poikilocytosis and anisocytosis were present as before.

On the same day the *liquor arsenicalis* was commenced, and improvement was immediate. The diarrhœa cleared up, the colour rapidly became more normal, and the child more responsive and bright.

Ten days later, on the 17th December, 1916, the blood count showed the following figures:—

Red Cells (dupl.)	2,300,000
Hæmoglobin	50%
Leucocytes (dupl.)	22,000
Colour Index	1

A differential count was not made.

Ten days later, on the 27th December, 1916, the count was:

Red Cells	3,300,000
Hæmoglobin	90%
Leucocytes	12,000
Colour Index	1.3

Many poikilocytes and microcytes and some polychromatophilia were present. No nucleated cells were found.

Differential count:—

Polymorpho-nuclear Cells	56%
Mononuclear Cells	37%
Eosinophile Cells	4%
Transitional Cells	3%

Some time later, on the 5th February, 1917, the child was re-admitted with suspected diphtheria, which turned out to be bronchitis, and the arsenic was suspended for a while and subsequently re-started.

A blood count was made on the 8th March, 1917, and showed:—

Red Cells	3,700,000
Leucocytes	16,000
Hæmoglobin	Not Estimated

Differential count:—

Polymorpho-nuclear Cells	44%
Mononuclear Cells	48%
Eosinophile Cells	6%
Transitional Cells	2%

The appearance of the red cells was still abnormal. There was considerable poikilocytosis and anisocytosis, and some polychromatophilia. No nucleated cells were seen.

A blood count was made on the 12 April, 1917, and showed (Dr. Buckley):—

Red Cells	4,000,000
Hæmoglobin	70%
Leucocytes	15,000
Colour Index	0.9

Nothing abnormal was seen in the stained films.

The last count, on the 8th May, 1917, gave:—

Red Cells (dupl.)	4,200,000
Hæmoglobin	80%
Leucocytes	7,000
Colour Index	1

The red cells appeared normal. No nucleated cells were noted.

(Giemsa) Polymorpho-nuclear Cells	45%
Mononuclear Cells	55%

Summary of the Blood Changes.

The red cells and hæmoglobin in this case were very low, but recovered rapidly under arsenic. Until improvement began to occur the colour index was about unity. At no time was the index low. When the improvement began to take place the index rose to 1.3. This may possibly be explained by an increase in hæmoglobin while the cells were were still of the megalocyte type. The number of leucocytes fluctuated considerably. On admission they were 14,000, which is approximately normal for a child of that age. Twelve days later they were found to be lower, but the next count a week later showed an increase to 37,000. A week after the first administering of arsenic the leucocytes were 22,000, and thereafter the successive counts gave 12,000, 16,000, 15,000 and 7,000. The chief point of note about the leucocyte count was the high figure of 37,000 at the time the child was about at the worst phase. If the count had only been made at this time the diagnosis from leukæmia would have been difficult, especially as myelocyte type cells were present. Usually there was an excess of mononuclear cells, as is common in children, but at the time of the count of 37,000 the polymorpho-nuclears were 65%. The eosinophile cells were increased throughout the illness, varying from 4% to 6%. Myelocyte type cells were only noted on the occasion of the high leucocyte count, and these cells were not over 5%. Myelocyte type cells may be found in the blood in almost any type of marked leucocytosis.

The characteristics of the red cells were those of pernicious anæmia. Macrocytes, microcytes and poikilocytes were abundant. Polychromatophilia was present, and nucleated cells were a marked feature.

The number of nucleated cells is one of the most prominent features of the case. Many of these were hard to classify, but typical megaloblasts and normoblasts were present. I follow the teaching that it is the nucleus and not the size of the cell which distinguishes megaloblasts. The normoblast is a cell varying in size with a dark staining "solid" nucleus, which is often bi- or tri-partite. The megaloblast has a vesicular nucleus with definite chromatin threads. Indications of mitotic division were seen.

For a while after the case had definitely improved, and even when the red cells reached 3,700,000, macrocytes, microcytes and polychromatophilia were present, but now the blood appears quite normal.

No nucleated cells were found after the 27th December, 1916.

Diagnosis.

I purposely do not intend to say much on this point. There are, it seems to me, several possibilities. The case may be considered:—

(1) As a case of pernicious anæmia in an infant.
(2) As a secondary anæmia brought about by more or less concealed hæmorrhage from the bowel or from some intestinal toxæmia.
(3) As a case of Somma's *anaemia splenica* infection, otherwise known as *anaemia pseudo-leucanaemica* infection (von Jaksch), or splenic anæmia of infants (Hutcheson).

The plastic nature of the bone marrow in infants may justly, I think, be held to explain many of the peculiar features; unfortunately this very fact makes the diagnosis more difficult.

In an adult the marked changes in the shape of red cells, especially the numerous macrocytes and microcytes, relatively high colour index and the presence of megaloblasts would justify the straight-out diagnosis of pernicious anæmia. But in an infant it is equally possible that all these changes may be but the special type of reaction of the more or less immature bone marrow to causes which in adults would produce a straight-forward secondary type of anæmia.

There is no great evidence, however, of any cause sufficient to produce such a condition. There was some history of traces of blood in the stools at some date previous to the anæmia; there was also some gastro-enteritis; but

neither of these were in any way special or were unduly severe, and do not, in my opinion, justify the conclusion that either or both were the primary cause of the anæmia. The father was a painter, but had discontinued this work for some time before the child was born, and as the mother had no signs of lead poisoning, it is hard to see how an infant in arms could have been affected, even if there had been paint, etc., in the house, which we are informed there was not.

Somma's anæmia is a somewhat vaguely-defined condition in young children, in whom it is associated with enlargement of the spleen. We find an anæmia with a low colour index, associated with marked megaloblast reaction. As far as I can find, the grade of anæmia is not usually so great as in this case, and the colour index is invariably described as low.

In any case Somma's anæmia seems a rather ill-defined group of cases, and the matter has not been cleared by von Jaksch's subsequent and erroneous nomenclature.

The future history of this case will be an interesting one to follow, and I am still doing monthly blood counts.

LEFT-SIDED CAECUM AND ASCENDING COLON, WITH ABSENCE OF TRANSVERSE COLON.[1]

By **C. E. Corlette, M.D., Ch.M.,**
Surgeon, Sydney Hospital.

C.A., aet. 30 years, a Greek, had for years complained of various gastric symptoms and of constipation. He was of neurotic temperament, a bad sleeper, and was very subject to attacks of mental depression. The chief gastric symptom was flatulency after meals, but at times he had pain and excessive discomfort, relieved by washing out the stomach. Analysis of an Ewald test-meal gave the following somewhat unusual result: Amount withdrawn, 200 c.cm.; normal smell and appearance, total acidity, 91, acidity from free HCl 60. Therefore, both HCl and organic acidity were very high. It was provisionally regarded as a case of hyperacidity with much stasis. Subsequently, he was sent to Dr. J. G. Edwards for X-ray examination of the stomach and intestine after a bismuth meal. Dr. Edward's report was as follows: "Stomach dilated and situated very low in abdomen. Duodenum normal in relationships. No irregularity in outline of stomach or duodenum. Stomach empties within six hours. No pyloric stenosis. There is a peculiar abnormality of the intestine. The caecum and ascending colon are to the left of the mid-line and ascend almost vertically to the splenic flexure, there being an absence of a good deal of colon. There is some delay in the terminal ileum and colon."

Under the heading "Appendicitis in a Patient with Caecum and Ascending Colon on the Left Side" I reported what seems to have been an exactly similar abnormality in *The Medical Journal of Australia*, November 15, 1915 (Vol. II., p. 511). In the October number of *Surgery, Gynecology and Obstetrics* for the same year (Vol. XXI., p. 442, 1915), Dr. Joseph H. Schrup contributed an interesting article on "Left-Sided Appendicitis," in which he describes another case of this same abnormality, with an illustration reproducing the skiagram taken. If this be compared with the skiagram of my present case (see figure), it will be found that the pictures resemble one another very closely. In connexion with his case, Dr. Schrup instituted a search through the literature, and this revealed the fact that the abnormality must be an exceedingly rare one, for he was only able to collect two previously recorded instances of it, and these he quotes in his paper. And yet, notwithstanding its rarity, I find myself now recording my second case of the same abnormality within two years!

A skiagram of the condition, taken by Dr. J. G. Edwards, is reproduced herewith. The figure is self-explanatory.

A.B. Represents the Large Intestine from Ileo-caecal Valve to the Splenic Flexure (C.)
D. Descending Colon. F. Mid-line of Body.
E. Rectum. G. Left Hip-joint.

[1] Read at a Meeting of the New South Wales Branch of the British Medical Association on May 11, 1917.

Reviews.

ARTERIO-SCLEROSIS.

"Diseases of the Arteries, including Angina Pectoris,"[1] is the title of a book in two volumes by Sir Clifford Allbutt. "The various chapters have all been published before, some of them in substance, again and again." Hence the book "has rather the form of a string of dissertations than of an orderly monograph." The proofs were corrected for press in the early summer of 1914, when the war burst upon us. Publication was consequently postponed.

Part I. is devoted to arterio-sclerosis, and deals with the physics of the circulation; the viscosity of the blood; causes, symptoms, morbid anatomy, diagnosis and prognosis of arterio-sclerosis; arterio-sclerosis and the kidneys; cardio-sclerosis and myocardial values and the treatment of arterial disease. Every aspect of this great study is exhaustively treated, and the work, emanating from so illustrious an author, should be minutely read and digested by every physician and pathologist. The author protests against the prevalent view of arterio-sclerosis as a disease. This, he says, "is not pathology but necrology, and the error penetrates into and confuses all the therapeutics of the subject." He is disposed to adopt the name of athero-sclerosis in place of arterio-sclerosis. In dealing with blood pressure reactions he emphasizes the experiments of Elliott and others on the influence of stimulation of the splanchnic nerves not only in causing vaso-constriction by direct action on the vessels, but also in causing the adrenals to secrete

[1] Diseases of the Arteries, including Angina Pectoris, by Sir Clifford Allbutt, K.C.B., M.D., F.R.C.P.; Volumes I. and II., 1915. London: Macmillan & Co., Ltd.; Demy 8vo., pp. 534 and 559 respectively. Price, 30s. net.

more adrenin. As regards pressure gauging, the author indicates the fallacies incidental to external or indirect manometry. "The outcome of it all is that if we wish to have uniform data we must keep to the same instrument, the same adjustment, the same limb and part of limb, the same observer, and, I had almost run on to say, the same patient." Another important paragraph: "Arterial decay does not itself set up cardiac hypertrophy; the cases in which the heart is hypertrophied are cases of hyperpiesia or of renal disease, the arterio-sclerosis being not the cause of the high blood pressures, but their effect." The rôle of alcohol and tobacco in the causation of arterio-sclerosis is critically examined. Alcohol "is not a cause in eminent domain, but it is a potent ally of any other poison which may be in co-operation with it." Concerning nervous symptoms of arterio-sclerosis, vertigo "may mean anything from the little swim of atonic cephalic vessels to which neurasthenics are subject on stooping, or the passing giddiness of a dyspepsia, up to labyrinthine disease, or a small apoplexy. . . . Labyrinthine vertigo, though usually, is not necessarily attended with deafness, nor even with tinnitus."

In Volume 2 the diagnosis and prognosis of arterio-sclerosis are dealt with. It is stated that it may be a matter of some difficulty to distinguish between hyperpiesia in its earlier stages and neurasthenia. As to prognosis, "In hyperpiesia, when dyspnœa has occurred, the malady has entered into its last stage; it is hardly remediable and quite incurable. The case will travel from bad to worse; the heart dilates, it is strung up with digitalis, it gets along again for a while, then relapses, is again perhaps pulled up, and so on; but it is a losing fight. Pulmonary œdema is menacing, and a whiff of it means imminent peril." In prognosis, the range of systolic pressures must not be relied upon too much. Besides emotional and other causes of temporarily exalted pressures, it is possible that individuals differ normally in their blood pressures. In treatment, as regards special dietaries, "temperance rather than exclusiveness should be observed, especially, it is supposed, in respect of the nitrogenous, and of those which contain purins; but really of the effects of purins we know little." "A milk diet, exclusively speaking, can be but a temporary resource—say for a few days—unless the patient be kept in bed." As regards drug treatment the author advocates the use of the nitrites, with discernment. Sodium nitrite must not be combined with potassium iodide, nor with any oxidizing agent. Mercury, as blue pill or calomel, in fractional doses, is invaluable. "The general faith in the iodides as moderators of high arterial pressures is curiously robust and perennial, notwithstanding the default of any definite evidence of this virtue in them." The indications for and against the use of digitalis are explained.

Part 2, Section I., is devoted to aortitis, including gonorrhœal, syphilitic and malarious aortitis and tuberculosis of the aorta.

Section II. embraces angina pectoris. Mock anginas are subjected to caustic criticism, and the author agrees with Mackenzie that the term pseudo-angina pectoris is useless and misleading, and should be eliminated from medical literature. In reference to tobacco angina the author states: "The genuine spectral horror or awe of angina I have never observed in mere tobacco angina, but reasonable alarm only. The vertigo, so frequent in tobacco excess, is often in part a cause of the alarm." The causes of angina are dealt with under the headings of remoter causes (age, station of life, sex and season) and proximate causes (bodily labour, the strain of continuously high blood pressure, mental and emotional causes, infections—gout, syphilis, malaria, rheumatic fever, malignant endocarditis, etc.). The cardinal and crucial symptoms of angina are described as "(1) Peculiar and startling pain, usually sternal in seat, but, as it forces its path into contiguous segments of the spinal centres, above and below the last cervical and upper thoracic circuit, spreading thence into widening areas—into the left arm, or right arm, or both arms, the neck and parts even still more distant, and frequently attended or alternated with paræsthetic sensations; sometimes traversing the segmental centres from sensory to motor tracts, to set up paresis or cramp of corresponding muscles, or even an atrophy of them. (2) Arrest or restraint, brief but imperious, of respiration, and more or less of all muscular activity. (3) Sense of impending death, not constant, but in many cases appalling; often, even in slight or incipient cases an organic dread or sense of ill-omen as contrasted with rational apprehension." Accidental or subsidiary features include, amongst others, high arterial pressure, vaso-motor disorders and sudden death by cardiac inhibition. The following hypotheses as to the interpretation of angina are considered: (1) Distension of an enfeebled ventricle; (2) cramp or spasm of the heart, analogous to cramp of a voluntary muscle; (3) an "intermittent claudication"; (4) a myocardial ischæmia, generally due to an affection of the coronary arteries, this ischæmia being the direct cause of the pain; (5) neural origin; neuritis, neurosis, neuralgia or vaso-motor storm, local or general. The author's own interpretation should be carefully read, and particularly the pages on angina and aortitis and angina and aneurysm. Diagnosis and prognosis are adequately dealt with, and the work terminates with a chapter on treatment. The heart must be protected against inhibitive shock. This is best attained by the use of atropine. Gout, if associated with angina, must be treated. Amongst other methods of treatment fully described are high frequency current, rest, diet, drugs, including iodides, salvarsan (in syphilitic cases), arsenic, bromides, nitrites, theobromine, and, during an attack, somnoform, oxygen inhalations, amyl nitrite and morphine.

Naval and Military.

The 313th list of casualties sustained by Australian troops abroad was issued on June 21, 1917. Under the heading "killed in action" there appears the name of Major Andrew Victor Honman (see *The Medical Journal of Australia*, June 16, 1917, page 510). From the same list we learn that Major Lionel Wilfred Bond and Captain Charles Herbert Leopold Leedman have been wounded. Both these officers have remained on duty. The 314th list of casualties, issued on June 22, 1917, does not contain the names of any medical men. The 315th list, which was issued on June 25, 1917, records that Captain Cedric Murray Samson has been severely wounded, and that Captain Ian Canute Gordon Glassford has been wounded. Among those ill in hospital are Captain Donald Ian Robertson Smith and Captain Sydney Stewart Shirlow.

We learn from the Hobart *Mercury* that Captain Herbert Percival Brownell, R.A.M.C., has been promoted to the rank of Major, and has been awarded the Distinguished Service Order for devotion to duty under heavy shell fire. Captain Brownell is a Melbourne graduate, and practised in Mitcham, South Australia, prior to joining the Royal Army Medical Corps.

News of Men at the Front.

Members are invited to contribute items of news of medical officers serving in the Australian Imperial Force abroad. We feel sure that abstracts from letters received from men at the front will be read with intense interest by their many friends, and therefore ask the recipients of these letters to assist us in making this column an attractive one. We are indebted to Dr. Archie Aspinall for the following notes:—

Dr. C. Wassall and Dr. W. E. Kay, who left Australia in 1914 as Captains attached to the First Field Ambulance, have now the rank of Lieutenant-Colonel, and are in charge of the 12th and 2nd Field Ambulances respectively.

Lieutenant-Colonel R. J. Millard has returned to England as Acting Director of Medical Services. He has recently been in temporary command of the No. 2 Australian General Hospital in France.

Captain Martel Aspinall has been transferred from the No. 14 Australian General Hospital to the 4th Light Horse Field Ambulance, and is now at the front relieving Major Vernon as Regimental Officer to the 11th Light Horse.

Lieutenant-Colonel Skipton Stacy is expected to reach Sydney early in July. Before leaving England, he spent several weeks visiting the London hospitals, and more especially the orthopædic hospitals.

The Medical Journal of Australia.

SATURDAY, JUNE 30, 1917.

Infantile Mortality.

The Committee appointed by the Department of Trade and Customs to enquire into the causes of death and invalidity in the Commonwealth has issued a report on infantile mortality. We propose to publish in a future issue a summary of the data on which the Committee build up their arguments, and to give a general outline of the contents of the report. The Committee have recognized the extreme importance of ascertaining the direct and indirect causes of death during infancy, and of combating these causes in the interests of the nation. The waste of healthy young lives in the great war is a colossal calamity which the Empire must face and attempt to remedy with all available means. Added to this waste of adult lives, there is the fact that the vast areas of the Commonwealth need a far larger population of workers to ensure its stability as a self-supporting component of the British Empire. The problem of the next generation rests primarily on a reduction of infantile death and infantile disease, and on the rearing of strong Australian children. This subject is a medical one, while the eugenic question of the increase of fertility is a social one. The Committee, in attacking the medical problem, find that over one-third of the deaths recorded of children under one year of age take place in the first week of life. They trace these deaths to inherited disease, of which syphilis is the most frequent and most important, and weakness, as well as to unhealthy environment and improper feeding. The remedies suggested, apart from those which have been recommended for the reduction of venereal infections, are largely educative. In the first place they advocate a system of ante-natal clinics, at which expectant mothers would receive skilled attention and advice. The essential lessons in elementary hygiene and the special instruction in the care of the infant should be given both to expectant mothers and also to growing girls. The Committee further suggest that these ante-natal clinics should be worked hand-in-hand with infant clinics, so that the health of the mother during her pregnancy and lactation, and of the infant before and after birth may be kept under expert supervision. In the next place, they regard with favour the plan of instituting home visitation of all mothers with young babies by expert women health visitors. They do not consider that the conditions at present obtaining in Australia justify the introduction of a Notification of Births Act, similar to the Act in existence in England and Wales, on account of the fact that, with a few exceptions, "the local authorities display a very perfunctory interest in the administration of health matters." Improved sanitary control of private dwellings, and a more rigid control of the milk supply are two essentials in the campaign aiming at the reduction of the infantile mortality. These recommendations are made with a laudable amount of confidence, and are unlikely to be challenged by anyone versed in the subject of infant care. The case is somewhat weakened by the suggestion that an organized attempt should be made "to apply existing knowledge and experience in one selected area, with the object of experimenting on a large scale to discover the most satisfactory methods of reducing the infantile mortality rate." It would seem as if the Committee had intended to apply the experiment throughout the length and breadth of the land, since they state that the success of their system could only be secured if the individual districts were inspired and controlled by the central health authority. The problem is not a local one, and success must depend on the application of knowledge to the whole community. If ever a strong case were made out for placing the control of health matters into the hands of one authority for the whole Commonwealth, it has been made out in the interests of infants.

While the suggestions of the Committee are excellent, and have proved themselves to be satisfactory in countries where they have been adopted, they are not without some danger traps, and they are not comprehensive enough to effect a great saving of infants. The Federal Statistician has shown that the wastage of infantile life is not only higher

in the first week of life than in any subsequent week, but is highest during the first day. Indefinite conditions, such as congenital weakness, marasmus and the like are probably unreal as important factors in this wastage. Statistics do not reveal how many healthy infants might be born alive were the practice of obstetrics more scientifically applied. The skilled obstetrician loses relatively few babies through a "difficult birth," but it is certain that very many infants die during birth as a result of inexpert management of the labour. The fault lies not so much with the alleged weakness or defect of the baby, as with the hand that guides too much or too little. In the next place, just as the average woman can be helped to bear a live infant, so nearly every woman can be helped to nurse her baby and keep it healthy. But this requires real expert guidance and not pseudo-science. The danger of the ordinary woman visitor, and we fear at times of the medical officer of the baby clinic, lies in the fact that persons with little or no training in a difficult specialty act as experts without any justification to the claim. We would therefore suggest as a preliminary to the excellent proposals of the Committee that a special training school for medical practitioners and nurses in the science and art of healthy baby rearing by real experts be established on a large scale.

SURGICAL PRACTICE IN NAVAL WARFARE.

In the planning of a great modern battleship, everything is sacrificed to purely naval ends. The shipbuilders are concerned with carrying space, stability, sea-worthiness and speed, but give no thought to the problems of the care of wounded sailors. Ventilation is admitted, provided that the system does not endanger the efficiency of the water-tight bulkheads. In the evolution of the modern battleship the medical officer selected what appeared to him to be the best-protected part of the ship for aggregating surgical dressings, and into the apartment chosen the wounded were brought for the purpose of receiving as much surgical treatment as the conditions admitted. Later, the naval authorities recognized the necessity of setting apart a portion of the ship for the so-called "medical distributing station," a curiously inapt term. As the size of ships increased the necessity of equipping two dressing-stations became apparent, and the authorities provided these below the water-line, as much out of harm's way as possible. Since these dressing-stations are still apartments in ships adapted after construction to their purpose, it is obvious that they are far from satisfactory, and the trials of the medical officers working below during and after an action at sea are considerable. Fleet-Surgeon C. L. W. Bunton, in the course of an engrossing article[1] on dressing-stations on large ships, pleads for a better design and for certain essentials, including adequate ventilation by inlet and outlet fans worked from within the dressing-station, fixed washing basins with waste pipes, and hot and cold water supply, and means for the removal of poisonous fumes, should an explosion occur between decks, among other things. It is quite obvious from the experience of the Battle of Jutland and of other naval engagements that the work of tending to wounded men aboard is a very urgent matter, and one which should have the consideration of the shipbuilder, as well as of the medical man. There is no reason why this essential part of a battleship should not be constructed with the same ingenuity as is displayed in the provision of other essentials. To place a dressing-station immediately next to the large uptakes from the stokeholds and boiler-rooms subjects patients to unfavourable conditions when severely wounded. In the planning of this department, it should be remembered that the severely wounded men will be transferred to a hospital ship or land hospital within twenty-four or thirty-six hours of the reception of the wounds under ordinary circumstances. In the Battle of Jutland the evacuation was delayed owing to the fact that several ships remained in the vicinity of the original action in the hope of cutting off some of the enemy ships from the base. The station is therefore required for the treatment of the patients during the period of intense shock. Fleet-Surgeon Bunton points out that the conditions are unfavourable to surgical intervention, and that the medical officer is well-advised if he does not attempt major operations or active treatment. His chief aims are to render the wounds aseptic, and to hand the patients over to better equipped hospitals in a condition for immediate operative treatment. The wounds are soiled by grease and dirt, but the high temperature of pieces of shell usually sterilizes the wound, and, by searing the tissues penetrated, arrests hæmorrhage. On this account he holds that there is no immediate necessity for the removal of these fragments. The treatment of shock kept him and his colleagues busy during the period following the termination of the Battle of Jutland. The difficulties involved may be gauged when it is remembered that the dressing-stations are overcrowded, ill-ventilated and over-heated, that the medical officers are working at utmost speed to attend to all the casualties, and that the stations are unsuited for surgical work. He found prompt infusion of saline solution imperative in shock associated with extensive hæmorrhage, while after grave injury or extensive burning adrenalin and pituitary extract frequently tided the patient over the critical period by raising his blood pressure. It is apparent that the metal of our naval medical officers can be depended on, in view of the remarkable success attending their efforts during and after the sea fights.

[1] *Journal of Royal Naval Medical Service*, April, 1917.

GOOD LIGHTING.

The human eye exercises its functions in consequence of the stimulation produced by the flux of light to the retina. The flux of light to the retina which may give rise to a visual sensation, varies over a wide range. The intensity of the light leading to the faintest sensation of vision is only one ten billionth of what falls on the retina in the blinding brightness of the sun reflected from a white surface. The sensibility of the retina at any moment is determined by the brilliancy of the field to which the eye is exposed. For any particular degree of illumination of the whole retina there exists a minimal brightness which elicits a sensation of light. A definite law connects the brightness of the field for which the sensitiveness of the retina has become adapted and the minimal amount of light that can be appreciated. The minimal brightness that can be seen is about one-thousandth of the field brightness when the retina is most sensitive, and diminishes with greater and less illumination of the whole retinal surface.

Good vision depends on the ability to distinguish detail in the objects viewed. It is not a function of a single variable, as is the case with the surface of a photographic plate. It is related to the brightness of the object presented to the eye to the brightness of the objects just previously seen and to the distribution of light on the surrounding objects. In any field of a given illumination certain differences in brightness exist which can just be perceived. The study of this power of discrimination reveals that the differences in brightness which can be appreciated with low intensities of light, are much less than at higher intensities, where there exists a considerable range of brightness, varying about 1,000 times, over which the power of discrimination is unaltered, while at somewhat higher intensities the acuity of vision diminishes.

The application of these facts to lighting is of importance. Four levels of lighting are found in practice: street or external lighting at night, with a brightness of 0.01 millilamberts; interior lighting at night, with a brightness of 0.1 millilamberts; interior lighting, with daylight, with a brightness of 10 millilamberts; and external daylight lighting of a brightness of 1,000 millilamberts. In street lighting the outlines of objects are alone required. Too great detail is unnecessary. As the sensitivity of the retina is low, harsh contrasts are needed. Glare must be avoided or the sensitiveness of the retina will be changed and dimly-lighted objects are not seen. In artificial lighting the arrangement of the source of light is of most importance, as a discrimination factor of half the maximum is needed for good vision. If the light is too concentrated, great contrasts will be produced, and objects in the shadows will be too dimly lit to give a sufficient discrimination. In interior daylight lighting attention should be paid to the direction of the light if the highest degree of vision is required. If light be too diffuse then the edges of small objects are no longer sharp, and the outline of fine detail is lost. Unidirectional light fulfils what is desired, while light from many directions leads to poor vision.

THE FRENCH-AUSTRALIAN LEAGUE OF HELP.

In December, 1914, a meeting was held in Sydney for the purpose of establishing an organization for the care of French soldiers and their dependents. Those present at this meeting formulated the objects of the French-Australian League of Help in such a manner that it would be enabled to co-operate with the French Red Cross Society in London and to join hands with any other society founded for the purpose of benefiting the soldiers of our Ally. Australia has given bounteously to the Belgian Red Cross Society and to other institutions for the relief of our Belgian Allies. The medical profession in the Commonwealth has sent valuable gifts to a fund for the assistance of the Belgian doctors. It is therefore fitting that the needs of France should not be forgotten, and in its turn France should hold our attention for a week in the capital city of New South Wales. Those who have had the privilege of serving the Empire in the fair fields of France have experienced the satisfaction of working side by side with French medical practitioners, and have given their best skill and energies to the treatment of wounded French soldiers. Those who remain in Australia from choice or necessity should be glad of an opportunity of assisting in maintaining the French Red Cross Society at its high level of efficiency. We feel sure that the sympathy of the medical profession with this wonderful organization will take a tangible shape during France's week, July 9 to July 14. The first event of the week is an attractive concert, organized by Mrs. Percy Bates, to be held at the Sydney Conservatorium Hall, at which several talented artists will perform.[1] Other musical performances will be given during the following days. By supporting the French-Australian League of Help to fill its coffers, we shall be supplementing in a small way the magnificent work that our colleagues on the western front are carrying out at the risk of their lives. It is little enough.

British Medical Association News.

SCIENTIFIC.

A meeting of the New South Wales Branch was held on May 25, 1917, at the B.M.A. Building, 30-34 Elizabeth Street, Sydney, Dr. R. Gordon Craig, the President, in the chair.

Dr. Burton Bradley read the notes of a case of *severe anaemia of "pernicious" type in an infant*, and exhibited a series of blood films under the microscope (see page 549).

Dr. W. F. Litchfield regarded the case as a very interesting one, and congratulated Dr. Bradley on the manner in which he had presented it. He stated that the fact that the child had got well was an argument against the diagnosis of pernicious anæmia. There were very few cases of pernicious anæmia in infants on record. In his own practice among children, of all the cases of primary anæmia he had seen only one that presented the characteristics of a true pernicious anæmia. The patient was a child aged two years and three months, and had been delicate from birth. There was a lemon-yellow anæmia. The spleen was

[1] Applications for tickets (2s. and 1s.) can be sent to the Editor of *The Medical Journal of Australia*.

not much enlarged. The red blood cells numbered 1,000,000, the white cells 12,000, the hæmoglobin value was 25%, and the colour index over 1. In the stained film myeloblasts, normoblasts, microcytes, megalocytes and poikilocytes were in evidence. The child died. He was inclined to believe that the case was one of true pernicious anæmia.

He suggested that Dr. Bradley's case might have been a secondary anæmia. It was not impossible that the anæmia may have depended on a syphilitic infection, more especially because syphilis often responded to arsenic. In von Jaksch's *anaemia infantum pseudo-leukaemica* the spleen was described as being enlarged, and often greatly enlarged, and there was a considerable increase in the leukococytes, especially the polymorphonuclear cells. There was also a tendency to considerable aggregation of myelocytes. Nucleated red cells were seen, and, in common with similar forms of anæmia, the embryonic types of cells were well represented. He did not think that Dr. Bradley's case was an instance of von Jaksch's disease. Although he would not commit himself, he was more inclined to regard it as a secondary anæmia.

Dr. Sydney Jamieson added his congratulations to those expressed by Dr. Litchfield. He agreed that there was no justification in considering the case one of anæmia caused by plumbism. There was no stippling of the red cells, or any other signs of lead poisoning. Moreover, he found that it was extremely uncommon for a pregnancy to reach maturity when the mother was under the influence of lead. Dr. Jamieson was very careful in regard to an expression of opinion concerning the diagnosis. All the features of a pernicious type of anæmia were present, and the improvement which had been observed while the patient was taking arsenic was also in favour of this diagnosis. On the other hand, the age of the patient was much against it. In pernicious anæmia there was a tendency towards recovery, and a still greater tendency towards relapse. It usually attacked persons over 30 years of age, and was more common in females than in males. Arsenic appeared to exercise a beneficial influence over the condition, and improvement not infrequently followed the use of antistreptococcic serum. There were two diseases which produced an anæmia not unlike that met with in Dr. Bradley's patient. These were malignant syphilis and cancer. He thought that it was a quite possible explanation of the case that the anæmia was due to a syphilitic infection.

Dr. F. Guy Griffiths considered Dr. Bradley's presentation of the case altogether admirable. He referred to a paper read by Dr. Camac Wilkinson in 1906 on pernicious anæmia. In the course of the discussion on this paper Dr. Litchfield had described a case very similar to Dr. Bradley's. He (the speaker) thought that it was a case of *pseudo-leukaemia infantum*. He admitted that the splenic anæmias in children formed rather an indefinite class of affection. Some clinicians doubted whether *pseudo-leukaemia infantum* was a definite disease, but the majority of the pathologists and hæmotologists dissented from this view. He had come to the conclusion that Dr. Bradley's case was an atypical instance of this disease. The patient presented all the appearances of it. The disease attacked infants between the ages of seven and twelve months. The pallor was of a lemon-yellow tint, and the little patients were usually comparatively plump. There was a striking enlargement of the spleen, and often some of the liver. The blood changes consisted in the marked diminution in the number of red cells and the presence of poikilocytosis and a striking leucocytosis, which was almost suggestive of leucocythæmia, and in the presence of megaloblasts and in the increased number of eosinophile cells. All these features were present in Dr. Bradley's case. In the next place, Dr. Griffiths gave his reason for coming to the conclusion that the case was not one of pernicious anæmia. It was not because of the age of the patient or the type of the blood change, but because there was a leucocytosis. Normally there were not more than 14,000 to 20,000 leucocytes per c.mm. In Dr. Bradley's case there was no lymphocytosis. The percentage of lymphocytes was actually lower than normal. The reverse was the case in pernicious anæmia. He did not regard improvement in response to arsenic as proof that the disease was pernicious anæmia. Arsenic was of use in many forms of anæmia, and improvement was often spontaneous in cases of pernicious anæmia.

Dr. R. Gordon Craig congratulated Dr. Bradley on the thoroughness with which he had carried out his observations. He regretted that it was still necessary for members who wished to demonstrate microscopical specimens to supply their own microscopes. He hoped that within a short time the Branch would be able to place a series of these instruments at the disposal of the members.

Dr. Burton Bradley, in his reply, said that the number of opinions expressed in regard to the diagnosis taught him that he was justified in refusing to commit himself. He admitted that syphilis could not be excluded merely on the ground that the mother's blood had given a negative response to the complement deviation test. He thought, however, that it was very unlikely that so young an infant would have syphilis, while its mother was apparently a perfectly healthy woman, and while there were no stigmata of the disease in either mother or child. He referred to the striking improvement which had taken place as the result of the arsenic medication. This fact had taught him a lesson, namely, that there was one other drug besides magnesium sulphate which was of use. An argument against the diagnosis of von Jaksch's disease was that the colour index was approximately 1 in his case, whereas in the disease mentioned it was said to be low.

Dr. Burton Bradley then gave a lantern demonstration on the microscopical diagnosis of *malignant condition of the uterus and adnexa*. He held that much advantage might be gained if members would do what he was attempting, that was to take up some section of their work, not necessarily the most difficult section, and review the result of their experience in relation to the work and requirements of other members. He pointed out that the clinical pathologist was in rather a transition stage. By a certain section of the profession he seemed to be regarded as something of a *prestidigateur*, who, armed with a microscope, section-cutter and so forth, performed a series of conjuring tricks with scraps of tissue or spots of blood, and settled the problem attacked. By another section he was regarded as something on the level of the lift attendant or chauffeur—a person to be kept in his place to hunt for tubercule bacilli and to report "yes" or "no" as to the presence of some condition or other expected by the clinician. The pathologist had himself to blame that the rest of the profession failed to realize that he was just one of the many specialists attacking one little corner of the great problem of sickness.

He maintained that clinical pathology could not be divorced from the other branches of medicine, and that the pathologist needed all the assistance that could be given by the clinician. Pathologists were not necessarily research men. They had a certain little field to cover with the aid of certain simple, if tedious, methods. He claimed that they were no more and no less scientific than the general practitioner who did his work well. The usual technique of clinical pathology was as simple almost as that of surgery.

The diagnosis of malignant disease of the female genital organs was of particular difficulty, and was not to be achieved easily by either the clinician or the pathologist. To render the work of the pathologist of value to the gynæcologist, it was essential that he should not work in the dark. He should be consulted and put in possession of all the facts of the case. After he had performed the tests required, he should be free to give an opinion in three ways, positive, negative or doubtful. There was often a difficulty in obtaining satisfactory specimens, and he laid stress on the fact that in the female pelvic organs the extraordinary and far-reaching changes occurring in connexion with the menstrual period and pregnancy were eternally intruding and complicating the problem. In other organs there was, as a rule, a relatively constant structural basis, which might be regarded as normal, and which could be utilized as a standard in measuring the degree of abnormality in any pathological variation. In uterus and adnexa they were dealing with structures which, during the child-bearing period of life, were practically never at rest, but always in a condition of preparation for, and recovery from, the periodic catamenia, except when interrupted by the greater preparation for, and involution following, pregnancy. It was rarely possible to obtain reliable information in regard to the exact stage of these physiological processes in the organs from which a pathological specimen was taken.

Dr. Bradley next dealt with the histological appearances of the lining ephithelium, tubular depression, interstitial tissue and muscular wall of the uterus under physiological conditions and in pregnancy. He referred to decidual cells in the scrapings from the uterus, which, in the absence of villi, had all the characteristics of an ephithelial new growth. The ovum in this case was in the tube.

In the next place he dealt with the difficulty of obtaining satisfactory specimens. This difficulty was a very real one, and was most unhappily associated with the factor of physiological variation. The specimens submitted usually consisted of (1) scrapings from the body of the uterus obtained by means of a curette, (2) snippings from the cervix, (3) portion of polypoid masses or of material expelled naturally from the uterus, and (4) portions of the organ submitted after operation or post mortem. He classified the first three types of specimens into: bad, worse, worst. It was at times impossible to say whence the specimen came, when it had been expelled from the organ. Snippings were frequently not properly taken. The usual mistake made was to take too small a fragment and not to give enough attention to the orientation. He referred to uselessness of superficial shavings of epithelium for diagnostic purposes.

Dr. Bradley projected microscopical sections on to the screen to illustrate the importance of ascertaining the exact situation of the tissue under examination. Scrapings had varying depth, and were often oblique. It was therefore common to see in the field structures derived from different levels of the organ. As an example he showed specimens including surface epithelium, glandular spaces and muscular fragments intermixed, when the intermixture was due to obliquity. The material satisfactory to the pathologist was unsatisfactory to the clinician and patient, for it was obtained after death or the loss of the organ. Polypoid masses were unsatisfactory, because in the presence of malignant disease they were often degenerated and unrecognizable. Uterine scrapings were usually of less positive value in the diagnosis of malignant disease. On the other hand, the finding of chorionic villi or decidual cells would offer an explanation for the symptoms.

In the next place he dealt with various malignant changes met with. Epithelioma of the cervix was usually recognized without difficulty from properly taken snippings. Carcinoma of the cervical glands were rare. Carcinoma of the body of the uterus was not specially differentiated from other carcinoma arising from columnar cell membranes.

Sarcoma was comparatively rare, but was at times secondary to myoma. He spoke of a case in which he had difficulty in determining whether the sarcoma had supervened on a myoma or whether the condition would be better interpreted as a type of cellular fibro-myoma. He expressed the opinion that it was difficult to be sure of a columnar-celled carcinoma from uterine scrapings, and that a guarded opinion should be given, even in apparently obvious cases. Epithelioma was usually recognizable in a satisfactory specimen, but the appearance of ingrowing epithelium at times associated with cervical lacerations, irregularities of the surface and faulty orientation was deceptive. An intense secondary inflammatory reaction could mask a growing neoplasm. There was rarely much difficulty in connexion with chorion epithelioma. Normal placental remains (villi and syncytia) should rarely cause confusion, but in sections of the wall of the uterus the syncytial masses deep in the muscle, as were met with in normal post-partum conditions, might be difficult to interpret.

True polypi were rarely suggestive of malignancy, but these at times contained irregular glands, suggesting a neoplasm.

In turning his attention to microscopical technique, Dr. Bradley spoke of the peculiarities of cell or nuclear structure, of irregular, multipolar, heterotype or homotype mitotic figures, and the importance of the study of these characters in the diagnosis of suspected malignancy. At times characteristic cells could be detected under a high power, while nothing abnormal was noted when the section was examined under low power. His experience had taught him that in the recognition of malignant and benign tumours it was essential to determine the characters of the individual normal cells of the organ, and to note the distinguishing structure of the abnormal tumour cells. High magnification was necessary for the recognition of atypical structure, staining, size and position of nucleus, type of chromatin and mitotic figures and of the relationship of these to other cells. When regarded separately, these changes might be very slight, but when they existed side by side they contributed to a definite picture. In dealing with the inflammatory changes met with in the uterine body, he pointed out that those met with in malignant and non-malignant affections of this organ did not differ materially from the inflammatory changes met with in other parts of the body. There was one point that had impressed itself upon his mind. This was the reversion of the interglandular specialized cell to the fibro-blastic type in certain conditions. He could not say exactly under what condition this occurred, but he assumed that it indicated a deterioration of special function and a return to a less specialized type of cell. Dr. Bradley concluded his remarks by exhibiting a number of microscopical pictures on the screen.

The discussion was postponed, owing to the late hour of the evening, until June 8, 1917, in order to allow Dr. W. F. Litchfield to read a paper.

At the meeting held on June 8, 1917, Dr. F. Guy Griffiths made some remarks on Dr. Bradley's paper. He wished to record his admiration of the philosophical tone which had been adopted. He considered that the necessity for candour and freedom of expression of opinion between the pathologist and the surgeon must be admitted. The pathologist should have the full knowledge of all the clinical facts. It was utterly preposterous to ask a pathologist for an opinion on a piece of tissue, without informing him whence it came and what the condition of the patient was. Dr. Griffiths also referred to the advantages of the *Balopticon* over the ordinary lantern. They had been able to recognize the various structures in the interesting microscopical sections with the greatest facility. He also expressed the opinion that it was not sufficiently recognized that there was no pathogonomic sign of malignancy. Some years ago Cathcart has written about the essential similarity of tumours. He had arranged a series of 30 to 40 bone tumours, varying from almost complete innocence to the highest grade of malignancy. It was obvious from this series that there was no dividing line between the benign and the malignant. Cathcart had stated that the same obtained in regard to tumours of other tissues.

Dr. R. Gordon Craig congratulated Dr. Bradley on his extremely valuable remarks. He considered that caution was needed in regard to the reports of pathologists, unless full information were accorded. He illustrated this by referring to conditions of the mouth in epulis of the gums. The pathologist would be led to diagnose sarcoma from the histological appearances only. Experience taught them that local removal of these growths was usually followed by complete cure.

Dr. Burton Bradley replied briefly.

Dr. W. F. Litchfield read a paper on Eric Pritchard's theory and treatment of rickets (see page 547).

Dr. Sydney Jamieson referred to Mosso's book on fatigue, in which the author dealt with fatigue as a toxæmia. Experiments on rabbits had shown that the fatigue produced by the over-stimulation of muscles could be immediately removed by washing out the veins.

Dr. Margaret Harper stated that she had treated rickets on the lines suggested by Dr. Pritchard with marked success. She agreed with Dr. Litchfield in his conclusions.

Dr. J. Burton Cleland, Principal Micro-Biologist of the New South Wales Department of Public Health, informs us that arrangements can be made for copies of his Annual Report for the year 1915 to be sent to anyone interested in the microscopical diagnosis of gynæcological affections. On September 8, 1916, Dr. Cleland demonstrated a large series of microscopical specimens of gynæcological cases before the New South Wales Branch (see *The Medical Journal of Australia*, October 14, 1916, page 328). In his Annual Report the full paper is published, together with photographic reproductions of some of the sections. We regret that the demand on our space has rendered it impos-

sible for us to have dealt with the 1915 Report of the Director-General of Public Health up to the present. We hope to publish an article on this Report and its appendices, including Dr. Cleland's, at an early date.

A meeting of the Council of the Victorian Branch was held at the Medical Society Hall, East Melbourne, on May 10, 1917, Professor R. J. A. Berry, the President, in the chair.

It was resolved that a Medical Agency be started under the auspices of the Medical Society of Victoria, and that Mr. C. Stanton Crouch be appointed Secretary.

A Sub-Committee was formed for the purpose of dealing with matters connected with the Agency. Dr. W. Kent Hughes, Dr. C. H. Mollison, and Dr. B. Crellin were appointed members of the Sub-Committee. Dr. Basil Kilvington was appointed to act in the place of Dr. Kent Hughes during his absence on military duty.

A communication from the Chairman of the Venereal Diseases Board was read, asking the Victorian Branch to nominate a representative to serve on the Board. Dr. C. H. Mollison was selected as the representative of the Branch for this purpose.

A meeting of the Council of the Victorian Branch was held at the Medical Society Hall, East Melbourne, on June 14, 1917.

It was resolved that a letter be sent to the Attorney-General of Victoria, requesting him to hold an enquiry into the conduct of the City Coroner, Dr. Cole, who, as reported in the daily press, had made unjustifiable remarks reflecting on the honour of Dr. Johnson Thwaites in connexion with the death of a woman in a private hospital, and asking him to take steps to prevent a repetition of the occurrence.

The action of the Elsternwick Dispensary in amending its rules in order to provide medical attendance to non-members was considered. It was pointed out that this action was in violation of the provisions of the Medical Societies Act. It was determined that the Council should act in conjunction with the Pharmaceutical Defence Limited in this matter.

The resolutions of the Federal Committee of the British Medical Association were considered. It was determined to adopt all the resolutions of the Federal Committee save three. These three resolutions were referred to the Legislative Committee for consideration and report.

The following were elected members of the Branch:—

Arthur Wadeson Le Souef, M.B., Ch.B., Melb. (1916), Broadmeadows Camp.

Alfred Raymond Fox, M.B., Ch.B., Melb. (1916), Stawell.

Colin Friend, M.B., Ch.B., Melb. (1916), Kew.

William Alexander Hamilton Birrell, M.B., Ch.M., Melb. (1915), Clifton Hill.

The Senate of the University of Melbourne, at its meeting held on June 19, 1917, adopted regulations relating to the endowment of a triennial lecture on some subject in medical science as a memorial to the late Captain Gordon Clunes McKay Mathison. A memorial tablet has been erected in the Melbourne Hospital, and the balance of the money subscribed for this purpose, which we understand amounts to £220, has been handed over to the Senate for the purpose mentioned. The Senate is to be congratulated on the wisdom of its choice of a memorial, although no triennial lecture is needed to keep Mathison's memory fresh in our minds.

Public Health.

THE HEALTH OF AUSTRALIA.

Infective Diseases Notified in Australia during the Quarter ending March 31, 1917.

	New South Wales.		Victoria.		Queensland.		South Australia.		Western Australia.		Tasmania.		Commonwealth.	
	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.[3]	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.[4]	Deaths.
Enteric Fever	577	48	266	34	292	27	128	18	102	20	68	5	1,433	152
Scarlatina	753	6	276	6	147	0	31	1	35	0	7	0	1,249	13
Diphtheria	1,616	76	947	40	460	23	229	18	181	8	155	4	3,588	169
Pulm'y Tuberc'losis	[1]349	231	431	190	124	83	166	93	95	60	53	24	—	682[2]
C'bro-Spinal Menin.	84	40	39	19	23	21	18	9	12	4	5	4	181	97
Poliomyelitis	8	1	8	1	6	0	0	0	0	0	0	0	22	2
Malaria	10	2	0	0	25	11	1	0	1	0	0	0	37	15
Puerperal Fever	—	37	—	14	5	2	5	5	—	4	2	3	—	65
Septicæmia	—	15	—	8	—	3	—	4	7	0	—	2	—	32
Bilharziosis	—	0	—	0	—	0	—	0	1	0	—	0	—	0
Pyæmia	—	1	—	1	—	1	—	0	1	0	—	2	—	5
Morbilli	—	12	—	8	—	7	196	0	—	5	—	0	—	32
Pertussis	—	61	—	24	—	6	412	14	—	11	—	2	—	118
Ophth. Neonatorum	—	0	—	0	—	0	—	0	5	0	3	0	—	0
Erysipelas	—	11	—	1	34	0	13	1	12	1	—	0	—	14
Ankylostomiasis	—	0	—	0	33	2	—	0	—	0	—	0	—	2
Beri-beri	—	—	—	—	—	—	—	—	2	—	—	—	—	—
Anthrax	—	—	—	—	—	—	—	—	1	—	—	—	—	—

[1] Notifiable only in portion of State.
[2] Including one death in Northern Territory.
[3] Exclusive of returns from one small district, which are not yet to hand. The total number of deaths from all causes in this district is given as 13.
[4] The total number of notifications are not given for those diseases which are not notifiable in all the States.

NEW SOUTH WALES.

The following notifications have been received by the Department of Public Health, New South Wales, during the week ending June 9, 1917:—

	Metropolitan Combined District.		Hunter River Combined District.		Rest of State.		Total.	
	Cs.	Dths.	Cs.	Dths.	Cs.	Dths.	Cs.	Dths.
Enteric Fever	3	0	0	0	3	0	6	0
Scarlatina	25	0	0	0	23	0	48	0
Diphtheria	57	2	2	0	109	4	168	6
*Pul. Tuberculosis	17	7	1	1	0	0	18	8

* Notifiable only in the Metropolitan and Hunter River Districts, and, since October 2, 1916, in the Blue Mountain Shire and Katoomba Municipality.

There have been three additional cases of variola at Brewarrina and 16 at Warren.

VICTORIA.

The following notifications have been received by the Board of Public Health, Victoria, during the week ending June 10, 1917:—

	Metropolitan.		Rest of State.		Total.	
	Cs.	Dths.	Cs.	Dths.	Cs.	Dths.
Diphtheria	53	1	47	2	100	3
Scarlatina	29	3	11	0	40	3
Enteric Fever	1	0	5	2	6	2
Pulmonary Tuberculosis	13	8	8	4	21	12
C'bro-Spinal Meningitis	2	—	0	—	2	—

QUEENSLAND.

The following notifications have been received by the Department of Public Health, Queensland, during the week ending June 9, 1917:—

Disease.	No. of Cases.
Pulmonary Tuberculosis	5
Diphtheria	29
Scarlatina	4
Erysipelas	2
Puerperal Fever	1
Enteric Fever	5
Ankylostomiasis	3
Malaria	6

SOUTH AUSTRALIA.

The following notifications have been received by the Central Board of Health, Adelaide, for the week ending June 2, 1917:—

	Adelaide.		Rest of State.		Totals.	
	Cs.	Dths.	Cs.	Dths.	Cs.	Dths.
Diphtheria	5	2	40	1	45	3
Pulmonary Tuberculosis	2	3	9	3	11	6
Scarlatina	1	0	6	0	7	0
Pertussis	0	0	6	0	6	0
Morbilli	0	0	4	0	4	0
C'bro-Spinal Meningitis	0	0	1	0	1	0
Enteric Fever	0	0	0	1	0	1

TASMANIA.

The following notifications have been received by the Department of Public Health, Tasmania, during the fortnight ending May 26, 1917:—

Disease.	Hobart. Cases.	Launceston. Cases.	Country. Cases.	Whole State. Cases.
Diphtheria	8	14	18	40
Enteric Fever	1	0	0	1
Pulmonary Tuberculosis	2	0	3	5
Scarlatina	1	0	0	1
C'bro-Spinal Meningitis	1	0	1	2
Puerperal Fever	0	1	0	1

Correspondence.

SEPARATION OF THE SYMPHYSIS PUBIS.

Sir,—The abstract on this subject in your issue of June 9 prompts me to record the only instance of this catastrophe occurring in my practice when in the country. An elderly primipara, small and wizened, was prematurely confined by me in the bush, without any intelligent assistance and amidst very unhygienic surroundings. The labour was unduly protracted, and, yielding to the importunities of the patient and bystanders, I terminated it under anæsthesia with forceps, a process at first difficult, but finally, after a sound like a muffled revolver-shot was heard, quite easy. There was an extensive laceration of the vestibule and vagina, reaching almost to the perineum, with the two halves of the *symphysis pubis*, separated by an interval of about 3.75 cm. (1½ in.), protruding through the anterior end of the wound. The urethra escaped injury. After delivery of the placenta, the pelvis was bound up and the patient removed to hospital. Here the lacerations were carefully sutured and the pelvis again tightly bound up. Septicæmia developed; the wounds broke down and sloughed; and the temperature reached 40° and 40.5° C. (104° and 105° F.), with very rapid pulse. This condition continued for some weeks with violent mania, during which the patient attacked her nurses and threw at them everything within her reach. She was of deeply religious habit, and in her delirium constantly proclaimed that everyone would think her unchaste because her infant was born less than nine months after her marriage. The child, I might mention, was small and of about 8½ months' gestation. Eventually the patient completely recovered. The symphysis united by fibrous union, and locomotion was not interfered with. Since leaving the country I was informed that the patient had been safely delivered of a child without untoward incident.

Yours, etc.,
JOHN MacPHERSON.

Macquarie Street, Sydney,
June 13, 1917.

THE TREATMENT OF CARBUNCLE.

Sir,—In the correspondence in your columns recently regarding the treatment of carbuncle, a most valuable line of treatment has apparently not been mentioned. That is the injection of an autogenous vaccine, or, failing that, a vaccine of mixed *staphylococcus albus* and *s. aureus*. Two years' experience of this in military camps and hospitals has convinced me that we have no means at our disposal so powerful as an adjunct to the local treatment, which has been so ably and clearly laid down.

Yours, etc.,
J. T. MITCHELL, M.D.

Central Flying School, Laverton,
Victoria, June 11, 1917.

THE METRIC SYSTEM IN PRESCRIBING.

Sir,—The adoption of the metric system for prescribing is logical, no doubt, and seems to be generally admitted as desirable. The means of ensuring its adoption is the crux.

Dr. Guy Griffiths' suggestions as to becoming able to think metrically are helpful and valuable.

Dr. J. C. Verco has had prepared and distributed to many South Australian practitioners a waistcoat pocket handbook of the preparations of the British Pharmacopœia with metric doses. This should prove of practical assistance to the beginner in metric prescribing.

Reforms, however, are seldom suddenly effected. The change at first must be optional in its application. "It is hard to teach an old dog new tricks." In spite of numerous statements to the contrary, it is hard for the average medico (and the majority are average) to think metrically and not according to the Imperial standard, with which their minds have been associated since infancy, and in which all their later training has been directed to deepening the mental impression.

If success is to be ensured we must strike at the root of the matter, and have our children taught the metric system and that only. The system must also be taught and practised in our Universities. I suggest that both systems be optional during the duration of the war and for two years after, to give our absent colleagues an opportunity of acquiring facility in the matter on their return to the Commonwealth.

In the meantime, the British Medical Association can approach the various State Governments and the Federal Parliament, and seek to have the metric system made compulsory in the State schools. A process of natural evolution would thus be commenced, and gradually and simply the new system become a nationally accomplished fact, not only in scientific circles but in business, mechanics, and the fine arts. This is a fine ideal for the British Medical Association to adopt as an outcome of war conditions.

For the present the part-time exponent of both systems, at his discretion, need not be unduly cast down by the thought of his mental limitations.

By prescribing the actual dose of the Imperial measure and making the chemist (who is usually an honest man and dependable) dispense the full quantity to the bottle size ℥iv., ℥viii., etc., fair approximation to accuracy can be obtained. There is no insuperable difficulty in adding 20 minims or 20 grains of a drug, as the case may be, to 3 quantities in a bottleful. The dose can be given in an ordinary Imperial measure glass, procurable for 6d., tough enough to withstand ordinary handling, and patients can be educated to their use. Until we have in vogue metric bottles and metric measure glasses we will only have approximation to accuracy.[1] This cannot be, it seems, till

[1] Vide I. C. Verco, *The Medical Journal of Australia*, July 1, 1916, page 3.

after the war. The difficulty of ʒ and ȝ symbol could be easily overcome by using Z for the ounce and A or H for the drachm symbol.

After all, medical men should write legibly, exercise care in putting a prescription on paper, and read it over twice, both with a view to accuracy and also ease of interpretation by the dispenser.

I write as a country general practitioner, who dispenses his own prescriptions, and also those of city practitioners.

The leaders in the profession must not forget that they are working for the common interest of all gradations, from the slow-thinking average man to the man of brilliant mentality environed by the best opportunity.

Yours, etc.,

SYDNEY M. VERCO,
M.B. et Ch.M. (Syd.).

Yankalilla, South Australia,
June 19, 1917.

Proceedings of the Australasian Medical Boards

VICTORIA.

The following have been registered under the provisions of Part I. of the "Medical Act, 1915," as duly qualified practitioners:—

Anderson, Joseph Ringland, M.B., Ch.B., Melb., 1917, 2 Highbury Grove, Kew.

Blaxland, Falkner John, M.B., 1911, Ch.M., 1912, Sydney, 105 Collins Street, Melbourne.

TASMANIA.

The undermentioned has been registered under the provisions of "The Medical Act, 1908," as a duly qualified medical practitioner:—

Park, Charles Leslie, M.B., Melb., 1907, D.P.H., Melb., 1915.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser," page xix.

Brisbane Hospital, Junior Resident Medical Officers.

Tumburumba Hospital, Medical Officer.

Medical Appointments.

IMPORTANT NOTICE.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429 Strand, London, W.C.

Branch.	APPOINTMENTS.
TASMANIA. (Hon. Sec., Bellerive, Tasmania.)	Medical Officers in all State-aided Hospitals in Tasmania.
VICTORIA. (Hon. Sec., Medical Society Hall, East Melbourne.)	Brunswick Medical Institute. Bendigo Medical Institute. Prahran United F.S. Dispensary. Australian Prudential Association Proprietary, Limited. National Provident Association. Life Insurance Company of Australia, Limited. Mutual National Provident Club.
QUEENSLAND. (Hon. Sec., B.M.A. Building, Adelaide Street, Brisbane.)	Medical Officers to the Selwyn Hospital, North Queensland. Brisbane United Friendly Society Institute. Warwick Hospital.
SOUTH AUSTRALIA. (Hon. Sec., 3 North Terrace, Adelaide.)	The F.S. Medical Assoc., Incorp., Adelaide.
WESTERN AUSTRALIA. (Hon. Sec., Health Department, Perth.)	All Contract Practice Appointments in Western Australia.
NEW SOUTH WALES. (Hon. Sec., 30-34 Elizabeth Street, Sydney.)	Australian Natives' Association. Balmain United F.S. Dispensary. Canterbury United F.S. Dispensary. Leichhardt and Petersham Dispensary. M.U. Oddfellows' Med. Inst., Elizabeth Street, Sydney. Marrickville United F.S. Dispensary. N.S.W. Ambulance Association and Transport Brigade. North Sydney United F.S. People's Prudential Benefit Society. Phœnix Mutual Provident Society. F.S. Lodges at Casino. F.S. Lodges at Lithgow. F.S. Lodges at Parramatta, Penrith, Auburn and Lidcombe. Newcastle Collieries — Killingworth, Seaham Nos. 1 and 2, West Wallsend.
NEW ZEALAND: WELLINGTON DIVISION. (Hon. Sec., Wellington.)	Friendly Society Lodges, Wellington, N.Z.

Diary for the Month.

July 3.—N.S.W. Branch, B.M.A., Council (Quarterly).
July 4.—Vic. Branch, B.M.A., Branch.
July 6.—Q. Branch, B.M.A., Branch.
July 10.—Tas. Branch, B.M.A., Council and Branch.
July 10.—N.S.W. Branch, B.M.A., Ethics Committee.
July 12.—Vic. Branch, B.M.A., Council.
July 13.—S. Aust. Branch, B.M.A., Council.
July 13.—N.S.W. Branch, B.M.A., Clinical.
July 17.—N.S.W. Branch, B.M.A., Executive and Finance Committee.
July 18.—W. Aust. Branch, B.M.A., Branch.
July 20.—Q. Branch, B.M.A., Council.
July 20.—Eastern Suburbs Med. Assoc. (N.S.W.).
July 21.—Northern Suburbs Med. Assoc. (N.S.W.).
July 25.—Vic. Branch, B.M.A., Council.
July 26.—S. Aust. Branch, B.M.A., Branch.
July 27.—N.S.W. Branch, B.M.A., Branch.
July 31.—N.S.W. Branch, B.M.A., Medical Politics Committee; Organization and Science Committee.

EDITORIAL NOTICES.

Manuscripts forwarded to the office of this Journal cannot under any circumstances be returned.

Original articles forwarded for publication are understood to be offered to *The Medical Journal of Australia* alone, unless the contrary be stated.

All communications should be addressed to "The Editor," *The Medical Journal of Australia*, B.M.A. Building, 30-34 Elizabeth Street, Sydney, New South Wales.

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Moisture	3.2

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(Signed)

A. NORMAN LEEMING, M.B., B.S., Lond.,
Assistant Bacteriologist to Guys Hospital.
E. P. MINNETT, M.D. (Brus.), M.R.C.S., L.R.C.P. (Lond.), Ph.C. (Lond.)

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LACTOGEN compared with COWS' MILK for INFANT FEEDING

When the supply of breast-milk for a Baby is insufficient in quantity and quality, or fails entirely, cows' milk alone is frequently used as a substitute. There are, however, many disadvantages attaching to its use, as compared with using **"Lactogen"** for this purpose.

COWS' MILK	"LACTOGEN"
COMPOSITION	
Unmodified cows' milk is by no means an ideal food for infants, for every animal specially secretes milk specially adapted to the requirements of its young, so that it becomes necessary to submit cows' milk to special treatment before it can be readily digested by infants. It is true that an approximation to the composition of human milk can be made by the addition of water, cream, and lactose to cows' milk, but this cannot be done very accurately in the ordinary household.	In the preparation of **"Lactogen"** the milk employed is carefully modified so as to resemble human milk in composition as closely as possible, and this being done on a large scale and under chemical control, can be very accurately performed.
PROTEID	
The proteid in cows' milk exists in a different condition to that in human milk, so that instead of curdling in a light flocculent manner as does the latter, it forms a tough leathery curd which very often proves difficult for a Baby to digest, and causes what is known as curd indigestion.	The proteid matter in **"Lactogen"** has undergone an alteration which makes it curdle in the same manner as in the case of human milk, and consequently it is very readily digestible.
FAT	
The fatty globules in cows' milk are larger than in human milk, with the result that the use of cows' milk has a tendency in some instances to cause fat indigestion.	In the manufacture of **"Lactogen"** the fat is subjected to an emulsifying process which reduces the fat globules to a remarkably fine state of division (even smaller than in human milk), so that when **"Lactogen"** is fed there is no likelihood of any fat indigestion.
BACTERIA	
In using ordinary cows' milk there is a great risk of bacterial infection, and notwithstanding the greatest precautions it is impossible to procure this in anything like a sterile condition. In the great majority of cases, owing to the fact of the milk having been drawn many hours before use, a considerable bacterial increase has taken place, so that it then contains large numbers of micro-organisms, frequently including many undesirable varieties, and unless properly pasteurised it may also contain pathogenic bacteria. In this regard, Dr. Still (Common Diseases of Childhood, p. 47) states that "Not "only throughout infancy, but through the whole period of "childhood the use of raw milk is attended with very serious "danger, a danger which in my opinion altogether outweighs "the disadvantages of heating the milk by pasteurisation, "boiling or sterilisation."	**"Lactogen"** is a sterile preparation, entirely free from all objectionable and pathogenic micro-organisms, and hence its use is a safe-guard against all milk-borne infective disease, especially infantile diarrhoea, which is so serious a cause of mortality during the summer months, when cows' milk frequently contains an excessive number of bacteria.
SCORBUTIC PROPERTIES	
The sole advantage apparent in the use of raw cows' milk is that it contains certain anti-scorbutic principles of an unknown nature. Dr. Still, however, points out that the anti-scorbutic power of fresh unboiled cows' milk is but slight, and, from the rarity of scurvy in infants fed on boiled milk, that it would seem but little scorbutic tendency occurs in milk, which has been merely heated to boiling point for a few seconds, as commonly done.	Though no complaint on the score of scurviness has ever been known from the use of **"Lactogen"**; to further avoid any risk of the production of infantile scurvy, every tin bears directions that a small quantity of fruit juice should be regularly given to all babies over the age of three months. In the preparation of **"Lactogen,"** the milk used is not subjected to any unnecessary heating, and that no loss of nutritive value is incurred by the action of heat on milk is demonstrated by Dr. Janet Clayton's Report to the Local Government Board (1912), in which, after reviewing all the available data, the conclusion arrived at is that "such small differences as have been found in the nutritive values of raw and boiled milk have been in favour of the latter."

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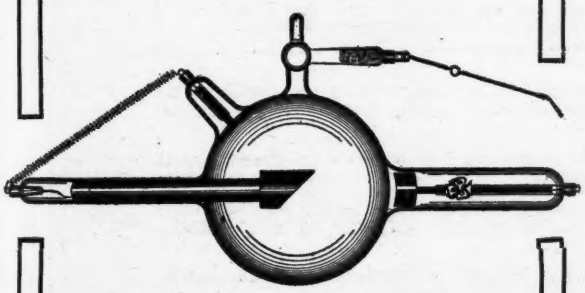

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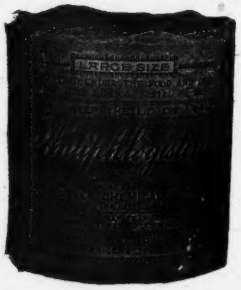

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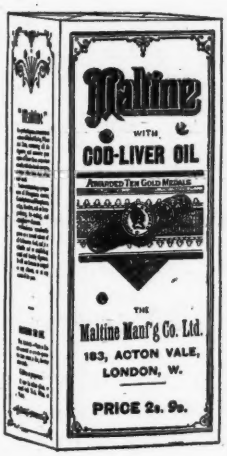

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